Chapter 6

Cardio-Oncology: Cardiovascular Risk and Surveillance in Cancer Therapy

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Abstract

Cardio-oncology has emerged as a critical interdisciplinary field at the intersection of cardiology and oncology, addressing the growing need for cardiovascular care in patients undergoing cancer therapy. With the advent of more effective oncologic treatments, cancer survival rates have improved substantially; however, this progress has been accompanied by a notable rise in treatment-related cardiovascular complications. Agents such as anthracyclines, HER2 inhibitors, immune checkpoint inhibitors, and radiotherapy are known to contribute to a spectrum of cardiovascular toxicities, including heart failure, arrhythmias, ischemia, myocarditis, and thromboembolic events.

The early identification and continuous monitoring of cardiovascular risk in cancer patients are essential for preventing irreversible cardiac damage and ensuring optimal therapeutic outcomes. Risk stratification must begin before initiating cancer treatment, incorporating both traditional cardiovascular risk factors and cancer-specific considerations. Modern surveillance strategies involve baseline and serial evaluations through echocardiography, cardiac biomarkers (e.g., troponins, natriuretic peptides), and emerging imaging techniques such as cardiac MRI and strain imaging. Additionally, the integration of artificial intelligence and machine learning is increasingly being explored for predictive risk modeling and personalized monitoring.

Effective cardio-oncology care requires a multidisciplinary approach that aligns oncologic efficacy with cardiovascular safety. This entails close collaboration between cardiologists, oncologists, and other healthcare professionals to formulate individualized treatment plans, manage acute complications, and implement long-term surveillance protocols. As cancer therapies evolve, the scope of cardio-oncology will continue to expand, underscoring the necessity of proactive surveillance and risk mitigation strategies in this vulnerable patient population.

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https://doi.org/10.58830/ozgur.pub780.c3259

1. Introduction

Over the past several decades, the field of oncology has experienced significant advances in diagnostic methods, targeted therapeutics, and supportive care, leading to improved survival rates for many forms of cancer.[1] With greater numbers of patients surviving longer, attention has increasingly turned toward the long-term complications of cancer therapy. One of the most pressing concerns is the cardiovascular toxicity that can result from various cancer treatments, leading to a rapidly growing subspecialty known as cardio-oncology.[2]

Cardio-oncology aims to balance the benefits of cancer therapy against the potential harm to the cardiovascular system. This discipline integrates cardiologists, oncologists, pharmacists, nurses, and other healthcare professionals, working collaboratively to identify individuals at highest risk, mitigate cardiovascular events, and optimize long-term patient outcomes.[3] While anthracycline-related cardiotoxicity has been recognized for decades, newer targeted therapies and immune checkpoint inhibitors have introduced novel mechanisms of cardiovascular injury. Consequently, a comprehensive understanding of cardiovascular risk and evidence-based surveillance strategies is paramount to the success of modern cancer treatment regimens.[4]

The purpose of this chapter is to: (1) summarize the mechanisms by which cancer therapies impact the cardiovascular system, (2) highlight established and evolving risk factors for cardiotoxicity, (3) present guidelines for cardiovascular surveillance and monitoring, and (4) discuss strategies to prevent or mitigate adverse cardiovascular events. By identifying and managing these risks early, clinicians can improve both oncologic and cardiovascular outcomes, ensuring that life-saving cancer treatments do not come at the cost of irreversible cardiovascular disease.

2. Overview of Cardiotoxicities Associated with Cancer Therapy

Cardiotoxicities encompass a broad spectrum of complications, including left ventricular (LV) systolic dysfunction, heart failure, arrhythmias, hypertension, ischemic heart disease, pericardial disease, valvular dysfunction, and vascular events (such as thromboembolism).[4,5] The precise manifestation of cardiotoxicity often depends on the class of agent used, the specific mechanism of action, dose, treatment duration, and patient-related factors (e.g., age, comorbidities, and genetic predisposition).

2.1 Anthracycline-Induced Cardiotoxicity

Anthracyclines (e.g., doxorubicin, epirubicin) are among the most effective chemotherapeutic agents in the treatment of several malignancies, including breast cancer, lymphomas, and leukemias.[6] However, their use is limited by a well-characterized cardiotoxic profile. The primary mechanism involves the generation of reactive oxygen species (ROS) that damage cardiomyocytes and lead to a progressive decline in LV ejection fraction (LVEF). Anthracycline-induced cardiotoxicity can be classified as either acute (occurring during or shortly after infusion), early (within the first year of treatment), or late (years to decades following exposure).[7]

Clinically, anthracycline-related cardiomyopathy presents with impaired systolic function and features of congestive heart failure. This cardiotoxicity is often dose-dependent, with the cumulative lifetime dose being a critical risk factor. Furthermore, older age, pre-existing cardiovascular disease, radiation therapy to the chest, and the use of other potentially cardiotoxic therapies can amplify the risk.[8]

2.2 HER2-Targeted Therapies

Monoclonal antibodies such as trastuzumab and pertuzumab have revolutionized the treatment of HER2-positive breast cancer.[9] These agents interfere with the HER2 receptor's downstream signaling pathways critical for tumor cell proliferation. Despite their therapeutic efficacy, HER2-targeted therapies can induce a form of cardiotoxicity distinct from anthracycline-induced damage. The process is largely considered reversible upon discontinuation, suggesting a non-dose-dependent mechanism that alters cardiomyocyte survival pathways.[10]

While the risk of developing heart failure remains lower compared to anthracycline therapy, patients with pre-existing cardiovascular disease or those receiving concurrent anthracycline-based regimens require careful monitoring. Periodic assessment of LVEF via echocardiography or cardiac magnetic resonance (CMR) is recommended to ensure early detection of decline in systolic function.[11]

2.3 Tyrosine Kinase Inhibitors

A variety of tyrosine kinase inhibitors (TKIs), such as imatinib, dasatinib, nilotinib, and sorafenib, have emerged as targeted therapies for chronic myelogenous leukemia (CML), gastrointestinal stromal tumors (GIST), and other malignancies.[12] Although these agents primarily target abnormal signaling in malignant cells, their activity is not entirely tumor-specific. Off-

target effects on the cardiovascular system can include hypertension, left ventricular dysfunction, QT prolongation, and arterial thrombotic events.[13]

For instance, vascular endothelial growth factor (VEGF) inhibitors (e.g., sunitinib, bevacizumab when used in combination regimens) can induce endothelial dysfunction, leading to systemic hypertension, proteinuria, and an increased risk of thrombotic events.[14] Given these potentially severe cardiovascular complications, patients on TKIs often require close blood pressure control and periodic cardiac function assessment.

2.4 Immune Checkpoint Inhibitors

Immune checkpoint inhibitors (ICIs) such as nivolumab, pembrolizumab, and ipilimumab have transformed the management of melanoma, lung cancer, and other tumor types by potentiating the host's immune response against cancer cells.[15] However, the reactivation of immune mechanisms can also target healthy tissue, leading to autoimmune-like toxicities, collectively referred to as immune-related adverse events (irAEs). Cardiovascular irAEs can include myocarditis, pericarditis, arrhythmias, and vasculitis.[16]

Although immune-mediated myocarditis is relatively rare compared to other irAEs, it carries a high mortality rate and demands immediate recognition and treatment.[17] Early diagnosis can be challenging as symptoms may be nonspecific, and cardiac biomarkers (e.g., troponin) can be variably elevated. Cardiac MRI and endomyocardial biopsy may be required in complex cases to confirm the diagnosis.

2.5 Radiation-Induced Cardiovascular Disease

Radiation therapy remains a critical component of treatment for many thoracic malignancies, including Hodgkin lymphoma and breast cancer.[18] However, the benefits of radiation must be weighed against the long-term risk of radiation-induced cardiovascular disease. Radiation can injure all structures of the heart, leading to coronary artery disease, valvular disease, pericarditis, conduction abnormalities, and cardiomyopathy.[19]

Risk factors for radiation-induced cardiotoxicity include the total radiation dose, fractionation schedule, and the specific techniques employed (e.g., use of cardio-protective shielding or gating procedures). Modern radiation therapy techniques aim to limit cardiac exposure, but patients who received higher doses of radiation in the past require lifelong cardiac follow-up. The latency period for radiation-induced cardiovascular disease can span decades, emphasizing the need for sustained vigilance.[20]

Table 1. Common Cardiotoxicities Associated with Major Cancer Therapies

Therapeutic Class	Examples	Primary Cardiovascular Toxicities	Mechanism Highlights
Anthracyclines	Doxorubicin, Epirubicin	LV dysfunction, Heart failure, Arrhythmias	ROS generation, oxidative stress, dose-dependent injury
HER2-Targeted Therapies	Trastuzumab, Pertuzumab	LV dysfunction (often reversible), Heart failure	Inhibition of HER2 signaling pathways in cardiomyocytes
Tyrosine Kinase Inhibitors	Imatinib, Sunitinib, Sorafenib	Hypertension, LV dysfunction, QT prolongation	Off-target effects on signaling pathways, endothelial dysfunction
Immune Checkpoint Inhibitors	Nivolumab, Pembrolizumab, Ipilimumab	Myocarditis, Pericarditis, Arrhythmias, Vasculitis	Autoimmune inflammation of cardiac tissue
Radiation Therapy (Chest Area)	Radiotherapy for Hodgkin Lymphoma, Breast Cancer	Coronary artery disease, Valvular disease, Pericarditis	Direct damage to coronary vessels, valves, and myocardium

Abbreviations: LV = Left Ventricular; ROS = Reactive Oxygen Species

3. Risk Factors for Cardiotoxicity

Cancer therapy–related cardiotoxicity is multifactorial, involving patient-specific factors, treatment-related aspects, and underlying comorbidities. Early identification of high-risk patients enables clinicians to implement protective strategies, including closer surveillance and prophylactic treatments.

3.1 Patient-Specific Risk Factors

1. **Age**: Extremes of age (children and older adults) are generally more vulnerable to cardiotoxic effects. Children treated with anthracyclines may develop subclinical cardiac dysfunction that manifests as heart failure in adulthood.[7] Older adults often have pre-existing comorbidities and may also have reduced cardiac reserve.
2. **Pre-existing Cardiovascular Disease**: Hypertension, coronary artery disease, valvular disease, and heart failure significantly increase

the risk of cardiotoxic events when combined with chemotherapy or radiation.[21]

3. **Genetic Factors**: Genetic variants in the enzymes responsible for drug metabolism and oxidative stress pathways can predispose certain individuals to cardiomyocyte injury.[22]
4. **Lifestyle Factors**: Smoking, alcohol use, obesity, and sedentary lifestyle can amplify the harmful effects of cancer treatments on the cardiovascular system.[23]

3.2 Treatment-Related Risk Factors

1. **Cumulative Dose**: Anthracyclines exhibit a well-known dose-dependent cardiotoxic effect, with a higher lifetime cumulative dose increasing the likelihood of developing heart failure.[6]
2. **Concurrent or Sequential Therapy**: The combined use of potentially cardiotoxic agents (e.g., anthracyclines with HER2-targeted therapies) exacerbates cardiac injury.[11]
3. **Dose Intensity and Schedule**: More intense chemotherapy regimens and shorter intervals between doses may escalate cardiotoxicity risk.[24]
4. **Radiation Field and Dose**: Radiation-induced cardiac damage is associated with both the total dose and the volume of cardiac tissue exposed.[20]

Table 2. Key Risk Factors for Cancer Therapy-Related Cardiotoxicity

Risk Factor Category	Specific Factors	Impact on Cardiotoxic Risk
Patient-Related Factors	- Advanced age- Pediatric age- Pre-existing CVD- Genetic predispositions- Lifestyle (smoking, obesity, inactivity)	Reduced cardiac reserve in older/pediatric patientsHeightened vulnerability in those with baseline heart diseasePotential for increased ROS susceptibility
Treatment-Related Factors	- High cumulative dose of anthracyclines- Concurrent use of cardiotoxic agents (e.g., anthracyclines + HER2 inhibitors)- Use of TKIs with known cardiovascular effects- Radiotherapy field and dose	Dose-dependent cardiomyocyte injurySynergistic toxicity when multiple agents are combinedEndothelial dysfunction and hypertension risks from TKIsLong-term radiation-induced structural damage
Medical Comorbidities	- Diabetes- Hypertension- Chronic kidney disease	Comorbidities amplify treatment-related cardiac stress
Oncologic Characteristics	- High tumor burden- Aggressive or advanced-stage disease	Often requires more intensive or prolonged therapy, increasing the likelihood of cardiotoxicity

Abbreviations: CVD = Cardiovascular Disease; TKI = Tyrosine Kinase Inhibitor; ROS = Reactive Oxygen Species

4. Surveillance Strategies

A key component of cardio-oncology is the systematic monitoring of patients for early signs of cardiotoxicity. Early detection and intervention can help mitigate irreversible damage and improve patient quality of life.

4.1 Baseline Cardiovascular Assessment

Before initiating cancer therapy with known cardiotoxic risk, a thorough baseline evaluation is essential. This includes a detailed medical history, physical examination, and assessment of cardiovascular risk factors. Laboratory tests should evaluate renal and liver function, lipid profile, fasting glucose, and, when indicated, cardiac biomarkers (e.g., troponin, natriuretic peptides).[25]

4.2 Imaging Modalities

1. **Transthoracic Echocardiography (TTE)**: Echocardiography remains the most common tool for assessing LV function due to its widespread availability, lack of radiation, and relatively low cost.[26] It

provides measures such as LVEF and diastolic function parameters. Strain imaging (particularly global longitudinal strain, GLS) has emerged as a sensitive marker for early changes in myocardial function.[27]

2. **Cardiac Magnetic Resonance (CMR)**: CMR offers superior tissue characterization and more reproducible quantitative measurements of ventricular volumes and function.[28] It can detect subclinical myocardial fibrosis and edema, especially in cases of suspected myocarditis from immune checkpoint inhibitors. Although cost and limited availability can be barriers, CMR may be indicated in equivocal echocardiographic findings or complex clinical scenarios.[29]
3. **Multigated Acquisition (MUGA) Scan**: Historically used to monitor LVEF in patients receiving potentially cardiotoxic chemotherapy, the MUGA scan provides accurate LVEF measurements but lacks the structural and functional details offered by echocardiography or CMR.[30]

4.3 Biomarkers

1. **Cardiac Troponin**: Elevated levels may indicate myocardial injury and can detect subclinical cardiotoxicity early in the treatment course, especially in anthracycline-based therapy.[31]
2. **B-type Natriuretic Peptides (BNP and NT-proBNP)**: These neurohormones increase in response to ventricular wall stretch and may serve as sensitive markers of cardiac dysfunction. However, they are not specific for chemotherapy-induced cardiotoxicity and can be elevated in other cardiac and non-cardiac conditions.[25]

4.4 Frequency and Duration of Surveillance

Guidelines from professional societies, including the American Heart Association (AHA) and the American Society of Clinical Oncology (ASCO), provide recommendations regarding the timing of surveillance imaging and biomarker assessment. For example, patients receiving high-dose anthracyclines or combination regimens may undergo imaging every 3 months during therapy and at 6-month intervals for at least 2 years after completion.[4,32] In contrast, those at lower risk may be monitored less frequently. Risk stratification models help tailor the surveillance intensity to individual patient profiles.

Table 3. Recommended Surveillance and Management Strategies

Phase of Care	Recommended Evaluations	Management/Intervention Highlights
Baseline (Pre-Therapy)	- Comprehensive history & physical exam- Echocardiography (LVEF, strain)- Biomarkers (troponin, BNP/NT-proBNP)- ECG	- Optimize modifiable risk factors (hypertension, lipids)- Consider prophylactic ACE inhibitor/beta-blocker in high-risk cases
During Therapy	- Periodic imaging (echo/CMR if indicated)- Regular biomarker measurements- Routine ECG monitoring (especially with QT-prolonging agents)	- Adjust or modify cancer regimen if early signs of cardiotoxicity develop- Manage hypertension promptly- Encourage lifestyle interventions
Post-Therapy (Follow-up Period)	- Long-term imaging follow-up (echo every 6–12 months in high-risk patients)- Ongoing biomarker assessment when indicated- Assessment for late radiation effects (if applicable)	- Continue or initiate heart failure therapies (ACE inhibitors, beta-blockers) if LVEF declines- Monitor and treat coronary artery disease and valvular pathology- Maintain regular exercise and cardiac risk factor control

5. Preventive and Protective Strategies

5.1 Pharmacological Interventions

1. **Neurohormonal Blockade**: Agents such as angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and beta-blockers have demonstrated efficacy in preventing or attenuating anthracycline-induced cardiotoxicity.[33] A prophylactic approach, starting these medications during cancer therapy in high-risk patients, may reduce the incidence and severity of LV dysfunction.

2. **Dexrazoxane**: This iron-chelating agent reduces free radical generation in the myocardium and is FDA-approved for the prevention of anthracycline-induced cardiotoxicity in select pediatric and adult populations with metastatic breast cancer who have reached high cumulative doses.[34] However, concerns about potential interference with chemotherapy efficacy have limited its widespread use. Current evidence suggests it is safe and does not significantly compromise oncologic outcomes.[35]

3. **Statins**: Emerging data indicate that statins may mitigate anthracycline-induced cardiotoxicity and reduce cardiac events in patients with pre-existing cardiovascular risk factors.[36] The cardioprotective mechanism

is thought to be related to statins' anti-inflammatory and antioxidative properties.

5.2 Modification of Risk Factors

Lifestyle modifications, including smoking cessation, regular exercise, weight management, and strict control of hypertension and hyperlipidemia, reduce the burden of cardiovascular disease in cancer survivors.[23] Coordination with primary care or a specialized cardio-oncology team can help patients adopt and maintain healthy behaviors throughout their cancer care trajectory.

5.3 Optimization of Oncologic Therapeutics

Oncologists and radiation oncologists can implement several strategies to mitigate cardiac injury:

1. **Use of Liposomal Anthracyclines**: Liposomal formulations (e.g., pegylated liposomal doxorubicin) minimize drug exposure to cardiomyocytes and lower cardiotoxicity risk while maintaining antitumor efficacy.[37]
2. **Adoption of Modern Radiation Techniques**: Intensity-modulated radiotherapy (IMRT), proton therapy, and deep inspiration breath hold (DIBH) techniques can limit cardiac dose and reduce long-term cardiovascular complications.[20]
3. **Algorithm-Based Decision Making**: By carefully selecting treatment regimens and scheduling based on patient risk factors, oncologists can balance therapeutic efficacy and toxicity. In some scenarios, avoiding concurrent use of anthracyclines and HER2 inhibitors or limiting cumulative drug doses may be considered.

6. Management of Cardiovascular Toxicities

Despite the best prevention strategies, some patients will develop cardiovascular complications that require immediate attention and management. A collaborative approach involving cardiology and oncology is essential.

6.1 Heart Failure and LV Dysfunction

- **Heart Failure Therapy**: Standard heart failure treatments, including beta-blockers, ACE inhibitors (or ARBs), and diuretics, form the cornerstone of cardiomyopathy management in cancer patients.[33] In

individuals experiencing anthracycline-induced cardiotoxicity, early initiation of these therapies can help restore or stabilize LVEF.[6]

- **Guideline-Directed Medical Therapy (GDMT)**: Clinicians should follow standard heart failure guidelines, adapting them as necessary to accommodate ongoing or planned cancer treatment. Renin-angiotensin-aldosterone system (RAAS) inhibitors and beta-blockers have demonstrable benefit in improving outcomes.[38]
- **Device Therapy**: Implantable cardioverter-defibrillators (ICDs) and cardiac resynchronization therapy (CRT) may be considered in patients with severe LV dysfunction or arrhythmias, following standard indications.[5]

6.2 Arrhythmias

- **Atrial Fibrillation**: Common in cancer patients, particularly those with comorbid cardiovascular disease and those receiving TKIs. Management includes rate/rhythm control strategies, anticoagulation (when appropriate), and addressing reversible causes (e.g., electrolyte imbalances, hyperthyroidism).[39]
- **Ventricular Arrhythmias**: Some therapies (e.g., TKIs, anthracyclines) can predispose patients to ventricular tachyarrhythmias. These may require antiarrhythmic medications, ICD placement, or electrophysiological interventions in severe or refractory cases.[5]
- **QT Prolongation**: Frequent electrocardiographic (ECG) monitoring is critical for patients receiving drugs that can prolong the QT interval. Dose adjustments or alternative therapies may be necessary when prolonged QT is identified.[13]

6.3 Hypertension

- **Blood Pressure Control**: VEGF inhibitors and certain TKIs frequently induce hypertension, which can exacerbate cardiac stress. First-line antihypertensive treatments generally include ACE inhibitors, ARBs, and/or calcium channel blockers, titrated to maintain blood pressure within guideline-recommended targets.[14]
- **Lifestyle Interventions**: Concurrent dietary modifications and exercise are encouraged for improved blood pressure control. Routine monitoring and prompt management are crucial to prevent end-organ damage.[23]

6.4 Ischemic Heart Disease

- **Coronary Artery Disease (CAD)**: Patients with chest radiation or systemic therapies that accelerate atherosclerosis are at high risk for CAD. Standard treatments, including anti-platelet therapy, statins, and revascularization procedures (e.g., percutaneous coronary intervention or coronary artery bypass graft), should be tailored to each patient's overall clinical condition and oncologic status.[5]
- **Stress Testing**: Noninvasive imaging stress tests help diagnose significant coronary artery stenoses and guide management.[21]

6.5 Pericardial Disease

- **Pericarditis and Pericardial Effusion**: Radiation, immune checkpoint inhibitors, and certain chemotherapeutic agents can induce inflammation of the pericardium. Management typically involves anti-inflammatory treatments (e.g., NSAIDs, colchicine) and monitoring for potential cardiac tamponade.[16]

7. Special Populations

7.1 Pediatric Cancer Survivors

Children treated for cancer during early developmental stages are at an elevated risk for late cardiovascular complications due to the cumulative toxicity of anthracyclines and radiation on the developing heart.[40] Long-term follow-up programs focus on regular echocardiographic evaluations, biomarker testing, and monitoring for modifiable cardiovascular risk factors (e.g., obesity and hypertension). The Children's Oncology Group (COG) provides specific guidelines for cardiac screening and follow-up intervals.[7]

7.2 Older Adults

Geriatric patients with cancer often have multiple comorbidities and reduced physiological reserve. Comprehensive geriatric assessment and close collaboration between oncologists, cardiologists, and geriatricians help personalize treatment decisions, minimizing both cancer progression and cardiovascular risk.[21] Dose reductions or use of less cardiotoxic regimens may be required to balance efficacy and safety.

7.3 Patients with Pre-existing Heart Failure

Patients with baseline heart failure pose a significant challenge, as certain cancer therapies may exacerbate pre-existing systolic dysfunction.[5] Proactive

involvement of a cardio-oncology team, potentially modifying treatment regimens, and aggressive heart failure management are crucial to preventing decompensation.

8. Future Directions and Research Opportunities

8.1 Identification of Biomarkers and Genomic Predictors

The discovery of novel biomarkers and genomic signatures predictive of cardiotoxicity could revolutionize the field by enabling precision risk stratification. Ongoing trials are evaluating multi-omic approaches, including proteomics and metabolomics, to identify subclinical cardiac injury and individual susceptibility to toxicity.[22]

8.2 Advanced Imaging Techniques

Technological advances in imaging, such as myocardial strain imaging, parametric mapping in CMR (T1 and T2 mapping), and positron emission tomography (PET) with novel tracers, may further enhance our ability to detect subtle cardiac injury and differentiate among various types of myocardial damage (e.g., inflammatory, fibrotic, ischemic).[27,29]

8.3 Cardio-Oncology Clinical Trials

Large, multi-center randomized trials are needed to evaluate the efficacy of preventive strategies, including pharmacologic interventions (e.g., ACE inhibitors, beta-blockers, statins) and modern radiotherapy techniques. Such trials would refine evidence-based guidelines for surveillance, prophylaxis, and management of cardiotoxicities.[4]

8.4 Integration of Digital Health and Artificial Intelligence

Telemedicine, wearable devices, and artificial intelligence (AI)–driven analytics hold promise for personalized monitoring and early detection of cardiotoxicity. Remote patient monitoring can capture real-time changes in vital signs, heart rhythms, and activity levels, facilitating prompt intervention for developing cardiac events.[41] AI models can process large volumes of data, potentially identifying at-risk patients earlier than traditional clinical assessments.

9. Conclusion

The remarkable successes of modern oncology have extended the lives of millions of patients. However, these gains bring an imperative to protect the cardiovascular health of survivors. Cardio-oncology has emerged to bridge

the gap between life-saving cancer therapy and the prevention of adverse cardiac outcomes. By understanding the mechanisms of cardiotoxicity, recognizing individual patient risk factors, adhering to evidence-based surveillance protocols, and implementing preventive strategies, healthcare providers can significantly reduce the cardiovascular burden in patients receiving cancer treatment.

The future of cardio-oncology is promising. Advances in precision medicine, imaging technologies, and supportive care interventions will further refine our ability to identify, prevent, and manage cardiotoxicities. Ultimately, the goal is to ensure that every patient has the best possible chance of overcoming cancer without sacrificing long-term cardiovascular health.

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